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AGRICULTURAL MEMORANDUM NO 967.

Transmission of Aujeszky disease by airborne virus.

by J. Gloster.

INTRODUCTION

On 1st August 1979 Aujeszky disease (pseudorabies or mad itch) became a notifiable disease in Great Britain under the Aujeszky Disease of Swine Order. Under this order all clinical disease suspected as Aujeszky disease (AD) had to be reported to the Ministry of Agriculture Fisheries and Food (MAFF) and standstill regulations applied. Following confirmation of disease advice was given to the owner on how to combat disease on his premises and standstill regulations were lifted.

After consultation with the members of the pig industry the Minister of Agriculture decided in December 1982 that steps to eradicate the disease should be implemented (in a postal poll producers owning 74.3% of the pig population in Great Britain voted in favour of such a policy). The Pig Industry Levy Bill was first presented to the House of Commons on 23rd December 1982; the bill, which applies to Great Britain, provides for the raising of a levy by the Meat and Livestock Commission for the purpose of meeting costs incurred by MAFF for compensating owners for slaughtering pigs and for consequential losses suffered by the owners of pigs which are slaughtered. A more detailed account of the Levy Bill and the steps that MAFF are in the process of taking to eradicate Aujeszky disease (AD) are given in Appendix 1. MAFF Notifiable Disease Section (Tolworth) is responsible for the implementation of the eradication policy. Met 08

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Agriculture Section have been asked by them to assess the potential for airborne disease spread. This memorandum assembles the available information about AD in a form that is suitable for providing the basis of advice to MAFF. An account of disease incidence and its financial losses is given. This is followed by a description of AD and the methods by which it is transmitted. The evidence for airborne disease transmission is then investigated.

DISEASE INCIDENCE AND FINANCIAL LOSSES

The first report of AD in the scientific literature is given by Aujeszky (1902); the disease appeared firstly in cattle and subsequently in a dog and cat. Hanson (1954) suggested that disease was probably present in the United States of America as early as 1813.

The first reference to AD in England did not occur until 1953 (Done and Venn, 1953), although it had been reported from Northern Ireland in 1939 (Lamont and Shanks, 1939). Prior to 1973 AD was of little concern to the pig industry in this country; the number of cases confirmed yearly was of the order 1 to 5 and these were spread throughout England, with no cases being recorded in Scotland and very few in Wales. By 1973 the number of affected promises detected had increased to double figures, the increase being mainly confined to the eastern-side of the country where the pig population is densest. During 1980, 22 new outbreaks were recorded with many being concentrated in a small area in Suffolk (Cartwright, 1982). During 1981 and 1982 and there were 10 and 43 cases respectively and by the middle of February 1983 there were a further 4.

The trend towards an increase in confirmed outbreaks of AD as seen in Great Britain has also been seen in many other countries. There has been a marked increase in the recorded cases in most affected countries, in some cases to epidemic proportions eg Holland, France, Belgium and the USA. In Holland during the first

half of 1976 there was a 300% rise corresponding to about 300 outbreaks (Basinger, 1979).

It is hard to generalise about the financial losses involved because the consequences of disease are highly variable. Herds that are seriously affected can suffer the consequences of poor cash flow over many years. The direct and consequential losses incurred during one year in a 550 sow herd in Yorkshire were estimated to be close to £100,000 (Pig Farming, 1982).

THE DISEASE

AD is an acute and often fatal, predominately nervous condition of domestic and wild animals. Amongst those animals affected are pigs, cattle, sheep, dogs, cats, foxes, rats and turkeys (a comprehensive list is given by Gustafson, 1975). AD is caused by a virus of the Herpes group. Viral particles consist of three main components, a core ($\sim$ 77nm diameter), a capsid surround (105-110nm diameter) and an outer envelope ($\sim$ 150nm diameter) /Gustafson, 1975/. With the principal exception of pigs, the natural reservoir for infection, most animals are 'dead end' hosts - that is they die and do not excrete virus after infection. All age groups of pigs are susceptible to the disease and severity is related to the strain of virus and the age of the pigs; the more virulent the strain of virus and the younger the pig the greater the mortality. Within the first two weeks of life piglet mortality can range from 5% to 100%. By the fourth week of age many naturally infected pigs recover. Pigs up to five months old may show nervous and respiratory symptoms and mortality may reach 15%. Adult pigs rarely show acute disease, perhaps only anorexia; pregnant sows may abort or produce mummified foetuses or still births, the former up to two weeks after malaise is noted. Sows recover clinically but may remain latently infected and occasionally may have reduced fertility. Abortion, infertility and loss of production of recovering fatteners could be more important economically to the pig industry than the classic or dramatic piglet losses.

DISEASE TRANSMISSION

AD has been shown to be transmitted through a variety of routes. In general these may be divided into three catagories; introduction of virus through infected stock, non porcine carriers of infected material and by airborne virus (Baskerville, 1981; Cartwright, 1982; Gustafson, 1975; McFerran, 1982). The first two catagories are dealt with in this section and the last one is considered in more detail in the the next section.

INFECTED STOCK

Disease is most commonly introduced onto premises by latent carrier animals. Following the acute phase of disease, the virus can no longer be isolated from the usual sites but may still be present within the animal in a masked or latent form. Subsequently, despite the presence of circulating antibody this virus may be reactivated and excreted; it has been suggested that reactivation may occur during periods of stress. Field evidence has shown delays of up to 20 months prior to excretion of virus. In Northern Ireland disease spread has, on occasions, been shown to occur after the purchase of apparently-healthy breeding animals. Often the infected animal has been a gilt and, when she has farrowed, she and her litter have remained healthy while other litters in contact have become ill.

The introduction of infected semen can cause disease; boars can excrete virus in semen intermittently and transmission of virus to sows at service can occur, but the frequency is not known (Akkermans, 1963).

NON PORCINE CARRIERS OF INFECTED MATERIAL

There is strong circumstantial evidence to show that the movements of farm vehicles, delivery lorries and shared equipment can all act as vectors for disease transmission. (Cartwright 1982). Other circumstantial evidence suggests

that man may act as a carrier of virus (Cartwright 1982). AD has not been recorded in man.

Although rats, mice and foxes can become infected, it would seem that they play little part in the spread of disease. The only evidence available to suggest otherwise is that of Maes and others, (1979) who suggested that brown rats could have been involved in localised disease spread when they had consumed infected material and then infected pig feedstuff with their oro-nasal secretions.

A few workers, particularly those in Northern Ireland and the USA, have isolated AD virus from faecal material; the majority of other workers have failed to detect virus in faeces. No evidence has been found which suggests that disease transmission by slurry occurs.

The role that birds play in the spread of disease is uncertain. Laboratory experiments suggest the only way they could be involved in disease spread is as mechanical vectors. It is possible to speculate that in the field the birds could carry infected material from the infected premises to the pigs feeding area; in the case of the larger carrion eaters this might be by the removal of dead piglets or aborted material (Cartwright 1982).

AIRBORNE TRANSMISSION OF AD

Airborne spread of disease relies upon a complex chain of events. There are three main phases; firstly a period of virus excretion, secondly the virus has to be carried towards a susceptible host and finally the virus has to initiate infection. Information on all these factors is required in order to make an accurate assessment of the potential for airborne disease spread. This section reviews the evidence available in the literature concerning each of the three phases.

VIRUS EXCRETION

Virus may be released into the atmosphere by three major methods:-

(1) Direct exhalation of particles produced in the respiratory system.

(2) the break-up of infected material upon impact with the ground.

(3) the formation of particles containing virus upon the ground which became airborne by various means e.g. animal movements, turbulence, use of farm equipment.

Experience with other airborne diseases suggests that direct exhalation of infected particles into the air is likely to provide the major part of airborne virus concentrations. Consequently, although virus has been isolated from milk, vaginal secretions, intermittently in semen and occasionally in faeces these are thought to be of secondary importance to release through the respiratory system.

Several workers have detected virus in nasal and oral secretions for up to 20 days after infection with the largest amounts detected on days 2-9 (Shope, 1934); McFerran and Dow, 1964). The only quantitative evidence for airborne virus excretion is that of Donaldson and others (1983). 4 groups of 10 pigs, housed in looseboxes, were challenged internassaly with one of four virus strains (Standley /UK/, Flixton /UK/, NIA-2 /Northern Ireland/ and U298/81/Denmark/). Air samples were taken in the looseboxes with a Litton air sampler. Additionally individual pigs, infected with Flixton (UK) strain, were sampled within a chamber with a 3-stage May impinger and cyclone sampler. Virus was recovered throughout the seven day test period, the highest recoveries being obtained between 3 and 5 days past infection. On a daily basis the maximum amount excreted per pig per 24 hour period was log $10^{5.3}$ $TCID_{50}$ (Tissue culture infective doses, sufficient to infect half the samples). It is of interest to note that these quantities of virus released are similar to those of ruminants affected with Foot-and-Mouth disease (Sellers, 1971).

Following the acute phase of viral excretion the virus cannot normally be isolated in body secretions and excretions, but may be present in the pig (mainly

in the central nervous system) in latent form. Subsequently, however, this virus may be re-activated and excreted. No information on airborne virus excretion at this stage, as distinct from the initial accute stage, is available.

Mention has been made, in an earlier section, that the first visible signs of disease often occur when pregnant sows abort or produce mumified foetuses. No information on airborne virus release during this period is available.

CARRIAGE OF VIRUS TO A SUSCEPTIBLE HOST

Once airborne, the virus has to survive in the atmosphere for long enough to be carried by the wind to a susceptible host. If infection is to occur on a given farm downwind, the virus will have to remain viable and airborne for longer periods in light winds than in strong winds. However, even in light winds, say 3 m sec^{-1}, airborne particles will travel many kilometres ($\sim$ 10km) in 1 hour. Consequently, it is possible that virus which remains viable and airborne for only a short period of time is available to cause infection many kilometres downwind.

The viability of AD virus depends upon the air temperature, light level and pH to which it is exposed. The relative humidity of the air may also influence virus viability, but no evidence yet exists to support the idea. Solomkin and Tutushin (1956) found that virus exposed on hay, straw, sacks, twine, wooden boards, potatoes, bran and oats remained viable for days; in winter (average temp -8°C) it survived for 35-46 days, in summer (average temperature 25°C) 10 to 30 days and in autumn (average temperature 14 to 18°C) 5-30 days. Kaplan and Vatter, 1959 found that 28% of infectivity survived after 5 hour at 44°C and Kaplan (Unpublished results) concluded that virus remained viable for weeks or months at -70°C. The relevance of these results to airborne virus is not understood.

Virus can be inactivated by exposure to ultraviolet light. There was a 90% reduction in virus viability when virus was exposed to 44 watt m^{-2} dose of

ultraviolet light irradiation for 40 secs at 2,500A (Kaplan, 1962). However, these levels far exceed those found at the surface at this wavelength, even on a clear summer's day.

Basinger (1979) states that the virus is relatively fragile and is susceptible to most disinfectants at low dilutions e.g. 2% FAM, 2% lysol, 2% citric acid.

The ability of virus to remain airborne is a function of the size of particle to which the virus is attached. Donaldson and others (1983) found that the association between infectivity and particle size was a function of the stage of disease (Table 1). It is interesting to note that there is a greater percentage of infectivity in the bottom stage of the impinger than for similar experiments performed with foot-and-mouth disease virus where the equivalent figure is 10-11% and Newcastle disease virus (3%) both of which are known to be transmitted through airborne virus. In a further experiment, Donaldson and others recovered high levels of infectivity when they exposed settling plates in the vicinity of affected animals. This indicates that a substantial quantity of infectivity is associated with larger particles, a finding which is consistent with their other experiments.

INFECTION OF A SUSCEPTIBLE HOST

For infection to occur, virus must penetrate susceptible sites on a animal in sufficient quantities and then be able to multiply. The factors which influence this are the route of infection and availability of virus. In addition, the larger the number of animals exposed to virus the greater the probability there is of infection.

ROUTE OF INFECTION

Infection has been shown to occur in the respiratory tract, alimentary canal and genital region (Saunders and Gustafson, 1964; Bitsch, 1975). Baskerville (1972) infected groups of 4 week old pigs by inoculating virus into their nostrils. The severity of the classical disease and the period between infection and death was directly related to the infecting dose of virus. The experiments showed that a dose of 10^1 $TCID_{50}$ could initiate disease.

Donaldson and others (1983) managed to infect a group of pigs housed in a different loosebox from a group of pigs excreting airborne virus. The two loose-boxes were connected by air ducting. The most probable part of entry for the virus into the second group of pigs was the respiratory system.

AVAILABLE CONCENTRATION OF VIRUS

Assuming that virus emitted into the atmosphere obeys the classical Gaussian description for an aerosol plume, the concentration at ground level downwind of a source can be derived (Pasquill, 1974). Blackall and Gloster (1981) developed a model which incorporated this theory to predict the downwind spread of foot-and-mouth disease. This model can also be used to calculate the downwind concentration of AD virus and hence an inhaled dosage by susceptible livestock can be deduced.

FIELD EVIDENCE FOR AIRBORNE INFECTION

Bitsch (1975) (a)

Bitsch presents the best documented evidence for airborne disease spread. The probable routes of infection in 29 outbreaks of AD in cattle between 1971 and 1973 were discussed in the light of epidemiological data and the histories of the individual outbreaks. The diseased cattle were divided into two separate groups; those which showed anterior puritus (itching towards the front end of the animal)

and those with posterior puritus (itching towards the rear end). The most likely route of infection in the former case was by the respiratory route and in the latter case the route of infection was much more complex (but included vaginal and alimentory infection and transmission by man).

In all cases attributed to respiratory infection, cattle were housed in the same building as infected pigs but separated from them by from several to a few tens of metres. Figure 1 shows the layout of the animal house in a typical outbreak. Piglets in two litters had died of AD a few days before the death of the cows. The house was low and aired through one ventilator in the ceiling just behind the two infected cows. Owing to the position of the ventilator the cows were the ones most likely to inhale virus emitted by the swine.

The author concluded that there were 12 cases in which airborne transmission of virus was the most likely reason for disease spread. Animals on nearly all the farms were housed in poorly or inadequately ventilated buildings. In two cases cattle were placed between the ventilators and the pigs. Nine outbreaks occurred in houses that were unsatisfactorily ventilated through simple air ducts and/or doors and windows. In three of these outbreaks the affected animals were very close or relatively close to swine, but in six outbreaks the affected animals, or most of them, were several meters away.

Bitsch (1975) (b)

Five outbreaks of AD in cattle were examined from the period 1974 and 1975. Evidence was presented which suggested that airborne infection was the most probable route for infection for at least one of the outbreaks; the conditions at this farm were similar to those described in Bitsch (1975)(a).

Anderson and Bitsch (1982)

These authors described the incidence of AD in Denmark during 1978 and 1979. Two distinct virus types were isolated during the period; one had a much higher pathogenicity than the other. The evidence presented supports the hypothesis that a highly pathogenic virus is capable of airborne disease spread.

An example of short distance disease spread from pigs to cattle is presented. In this case a group of affected fattening pigs were separated from a group of cattle by a wall; there were two windows with broken panes in the wall. The cattle were placed close to a ventilator and the air movements would have carried virus from the infected pigs directly over the subsequently affected cattle (Figure 2).

Two further cases, which involved disease transmission over much greater distances are also presented. In the first one a SPF (Specific Pathogen free) herd was infected about 6 months after it had been established. Buildings were new, and food was in special containers. Mice or rats had not been seen and introduction of the infection by persons seemed unlikely. AD had been diagnosed some years before in a herd with over 700 sows and 3,000 fattening pigs situated 500m away. A number of blood samples taken in this herd at the time of the outbreak in the SPF herd were all found to be serologically positive. Information about the weather during the period immediately prior to the outbreak did not exclude the possibility of an airborne spread from the previously affected herd. The authors concluded that with the amounts of virus that would have been produced by 3,000 acutely infected fattening pigs the risk of an airborne transmission to neighbouring herds was high.

The second case of disease transmission of some distance involved a closed breeding herd with over 200 sows which all became clinically affected. Approximately three weeks earlier an outbreak had started in a herd 125m away

with 150 sows and 200 fattening pigs. Wind directions during the acute outbreak in this herd supported the possibility of airborne transmission to the closed herd.

The authors concluded that it was noteworthy that in both the above cases where airborne transmission seemed likely, infection was considered to have been spread from herds with fattening pigs.

STARR (1979) and Basinger (1979)

An investingation into the route for spread of AD in an area of Suffolk (sometimes known as the 'Rishangles Triangle') was set up by MAFF. Starr was asked to supply meteorological advice to the leader of this project. Assuming that the real sequence of events was that of disease reporting and that meteorological observations taken 25 km to the southwest were representative for the area Starr showed that much, if not all, of the disease spread was consistent with airborne infection. However there is considerable speculation about the actual order of events in the area so no definite conclusions can be drawn from this work. It is of interest to note that animals here showed signs of respiratory symptoms.

DISCUSSION

From an epidemiological/meteorological viewpoint the airborne transmission of AD poses many unsolved questions. The ability of the virus to remain within the pig in latent (ie sub-clinical) form makes the re-construction of outbreaks very difficult; In addition the ability of certain virus strains to exhibit disease with low levels of pathogenicity means that disease may remain undetected for long periods. The outbreaks of AD in the 'Rishangles Triangle' (Starr, 1979) demonstrate these points; infection may well have remained latent for many months after being introduced into a premises or herd before the first reported case of disease occurred.

Alternatively, because of the mild symptoms associated with this virus strain, the actual disease may have been present but remained undetected. (Respiratory symptoms suggestive of rhinitis were observed and were readily attributed to that disease rather than AD). The only means of detecting presence of AD infection in some herds is by serological testing of pigs for AD antibodies. It may be assumed that presence of antibody means presence of infection, even though the virus may be latent.

Earlier it was suggested that stress may provide the 'trigger' which causes the recrudescence of virus excretion in latently infected pigs. Stress may be induced in an animal by various means eg. changes (particularly sudden or unusal changes) in the conditions to which the animal is exposed, poor levels of nutrition, transportation and mixing of unfamiliar stock. Pigs are generally fairly resistant to changes in climate and it is unlikely that these would often institute the required 'trigger' mechanism. The only evidence to indicate otherwise is supplied by Howarth (1969). He suggested that outbreaks were of a seasonal nature and followed periods of extreme weather; pigs which had passed from the acute to the latent stage of disease started to re-excrete virus and this infected others on the premises. If adverse enviromental conditions were combined with, for example, the movement of animals from one location to another, it is conceivable that the stress induced on the animal would provide the required stimulus for virus activation (for a discussion on the influence of environmental factors on pigs in transit see Allen and others (1974)).

Evidence from around the world suggests that different virus strains may behave rather differently; some virus strains associated with disease in Northern Ireland give mild respiratory infection whereas many of those found in Denmark give severe respiratory infection. It appears reasonable to suggest that where high levels of respiratory infection are present there is a greater potential for airborne disease spread. It is interesting to note that over the recent years, in many countries, the virus strains involved have tended towards giving more respiratory symptoms. At the same time the number of reported cases has also markedly increased.

Examples of disease transmission over a matter of a few metres or perhaps tens of metres have been quoted. In all such cases the number of animals excreting virus would have been low. The cases in which airborne disease has been estimated

to have travelled the furthest involved many affected animals. With the trend towards larger units or herds in the pig industry it is reasonable to assume that in future AD outbreaks, much larger virus sources will be present. The intensification of pigs, away from a virus source will also increase the risk of airborne disease spread over greater distances (Gloster and Field 1982).

Thus, the tendancy for virus strains to exhibit more respiratory symptoms (probably resulting in higher levels of virus excretion into the atmosphere) and the increase in numbers of potential excreters and receivers suggests that there will be a much greater risk of airborne disease spread in the future than ever before.

CONCLUSIONS

1. Strong evidence has been presented to show that airborne transmission of AD occurs under laboratory conditions.

2. Circumstantial evidence has been given supporting the hypothesis that airborne transmission occurs in the field.

3. The following questions must be answered before a satisfactory assessment of airborne disease spread can be made:-

   (a) How much virus, is produced by infected pigs and over what period does excretion occur in the field? - No quantitative measurements have yet been made from naturally infected animals.

   (b) What, if any, virus emissions into the atmosphere occur when sows abort or produce mummified piglets?

(c) What are the survival characteristics of airborne virus? - at the moment no measurement of virus viability has been made using airborne virus.

(d) How much virus is required to initiate infection? How does the amount differ for each of the potential routes?

ACKNOWLEDGEMENTS

The author wishes to thank Dr A I Donaldson, Animal Virus Research Institute, England and Dr A E Wrathall, Central Veterinary, Laboratory, MAFF, Weybridge, England for their help in writing this Memorandum.

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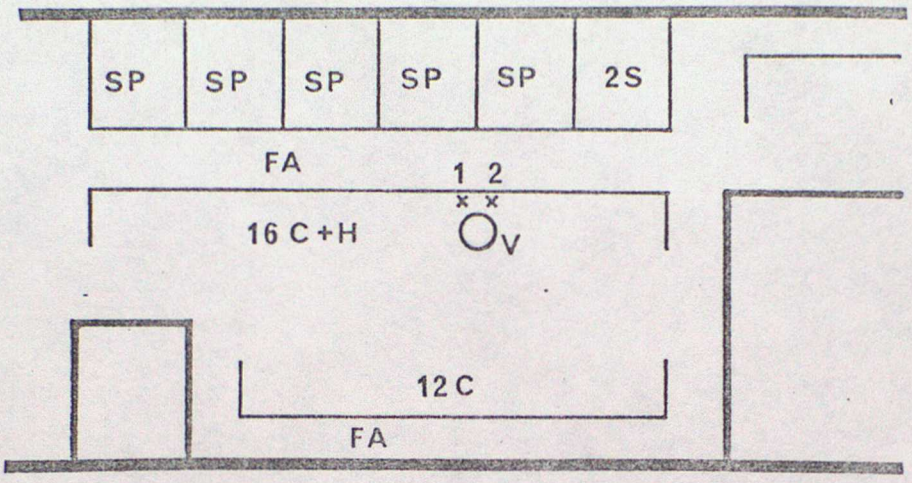
Any Comments or suggestions with regard to this memoradum should be addressed to the author, Meteorological Office, London Road, Bracknell, Berkshire.

March 1983.



C Cow

S Sows

SP Sows with sucklings

FA Feeding alley

V Ventilator

X Location of affected cattle

H Heifer

Figure 1 Plan of house showing location of animals.

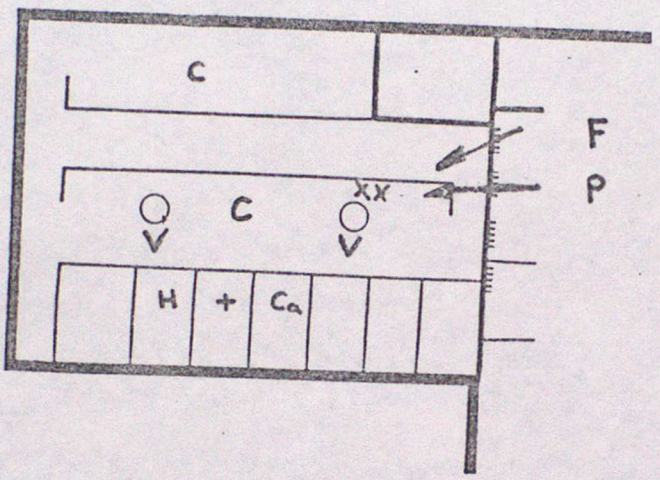


Fig. 2 Plan of a cowhouse, where Aujeszky's disease occurred in two cows

V ventilator

FP FATTENING PIGS

H Heifers

Ca Calves

C Cows

V Ventilator

X Affected animals

← Air currents from room with FP to cowhouse.

TABLE 1

Airborne AD virus recovered from pigs sampled individually using a 3-stage impinger

DAY POST INOCULATION	VIRUS RECOVERY*		
	T**	M	B
2	1.1 (31)‡	1.1 (31)	1.2 (38)
3	1.5 (47)	1.3 (29)	1.2 (24)
4	1.3 (58)	0.8 (18)	0.9 (24)

*Mean total virus ($\log_{10}TCID_{50}$) collected from 4 pigs in each stage of the impinger after 15 min. sampling period at 55 l min^{-1}

**T, M, B: top, middle and bottom stages of impinger (> 6 um, 3-6 um, < 3 um).

‡ percentage infectivity in each stage of the impinger

APPENDIX: Extract from Veterinary Record 5th February 1983, Vol. 112 Number 6.

Aujeszky's disease: 250 herd slaughter forecast

Up to 250 pig herds are expected to be slaughtered out during the forthcoming eradication programme for Aujeszky's disease. This forecast was given in the Commons on January 27 when the Pig Industry Levy Bill, which will enable the eradication scheme to be set up, went through all its stages after less than two hours debate.

Moving it, Mr Alick Buchanan-Smith, Minister of State for Agriculture, said that the disease was a viral infection primarily affecting pigs. The clinical signs were usually most apparent in sucking pigs and pregnant sows and piglets almost invariably died of the disease. It was spread mainly by movement of infected pigs, had been known to exist in Britain since 1953 and was of the greatest concern in areas where the pig population was concentrated, like the eastern counties.

If it was to be eliminated, it was essential that slaughter of infected herds was followed by disinfection. Vaccination was not an option.

The possibility of a control and eradication policy had been under discussion for some years. A major difficulty had been that under animal health legislation, the minister was bound to pay compensation for animals slaughtered for animal health purposes. Additional government funds were not available to meet such expenditure in the case of Aujeszky's disease.

The industry had voted for a scheme which would include a maximum compensation of £300 per animal slaughtered plus 20 per cent as compensation for breeders, weaners and fatteners raised as part of a breeder-fattener operation on a single premises or plus 5 per cent for slaughter of fattening pigs on separate premises.

While the government could not provide the compensation they would support eradication fully through the veterinary service and the facilities of the agriculture departments, said Mr Buchanan-Smith.

The cost of ministry staff involved, including work which the veterinary service undertook in its professional capacity, would be borne by the agriculture departments. They hoped to contain that cost within existing provisions.

The best estimate they could make was that 250 herds would have to be slaughtered, most of the work being undertaken during the first year of the campaign, subject to demands on veterinary manpower which might arise if there were outbreaks of other important diseases.

They would start on eradication as soon as they reasonably could, subject only to physical problems of getting the scheme into operation.

Mr Mark Hughes (Lab. Durham) endorsed the bill on behalf of the Opposition, although he regretted that such a scheme had not been started before the losses of recent years.

Winding up, Mr Buchanan-Smith said he would hate any feeling to emerge from the debate that the disease could be a problem for consumers of pigmeat or pig products.

Tackling eradication

Plans are well in hand as to how to tackle the disease once the green light is given to the State veterinary service. All isolated herds in areas where the disease is not prevalent will be dealt with first. Once the scheme comes into operation, movement restrictions will be applied to these herds, numbering in the region of 30 to 40, and testing and slaughter, where infection was confirmed, would soon follow.

In the three areas where the disease is concentrated, the 'Rishangles triangle' in East Anglia, Humberside and North Yorkshire, the ministry will approach the problem in three phases. In phase 1, those herds which have been recently affected and which exhibit clinical stages of the disease will be killed out and a 2 km Aujeszky's disease-free patrol area established around the farm. The farmer will not be able to restock until this patrol area has been established, and some problems are forecast in maintaining these areas where a number of closely neighbouring farms are affected. It is expected that this phase will take the shortest time to complete.

The second phase will involve the testing of known infected herds since 1979 (when the disease became notifiable), and where infection was confirmed, slaughtering of stock and the establishment of a patrol area. In the third phase the same approach would be adopted to those herds known to have been infected before 1979. In both these phases, the decision to confirm disease will rest with the ministry veterinary staff at Tolworth. The operation will broadly follow the standard procedures for the eradication of other diseases, although the ministry recognises that there would be no veterinary logic in slaughtering out a larger herd with one seropositive. Those pigs not clinically affected with disease and of marketable weight would be salvaged, involving possibly 400,000 pigs.

In all the phases priority would be given to weaner/breeder herds, with breeder fatteners next and pure fatteners last.

Mr Terry Heard, who has represented the BVA during negotiations with the ministry about how the scheme should run, foresaw a number of constraints that the ministry would be put under during its operation. First was the availability of manpower. There was also the difficulty of making available valuers to assess the worth of the stock, transportation and slaughtering facilities. Finally there was the depressing effect the extra amount of slaughtered pigs suddenly dumped on the pig market during the opening weeks might have.

On the financial side of the scheme, the NFU has set up a company to handle the levy money raised by the producers. Following slaughter of stock, the ministry would pay compensation 15 days after valuation and then would be reimbursed by the NFU company. All that the ministry would pay for would be the costs of veterinary manpower and laboratory testing. The cost of employing slaughtermen, valuers, disposal of carcases and compensation for feedingstuffs and other materials seized and destroyed will have to be met by the levy fund.